



## Newer insecticides for plant virus disease management

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### ABSTRACT

Effective management of insect and mite vectors of plant pathogens is of crucial importance to minimize vector-borne diseases in crops. Pesticides play an important role in managing vector populations by reducing the number of individuals that can acquire and transmit a virus, thereby potentially lowering disease incidence. Certain insecticides exhibit properties other than lethal toxicity that affect feeding behaviours or otherwise interfere with virus transmission. To evaluate the potential of various treatments against the *Bemisia tabaci*-transmitted *Cucurbit yellow stunting disorder virus* (CYSDV), insecticide field trials were conducted in Yuma, AZ, USA, during spring and autumn growing seasons. Differences in vector-intensity each season led to mixed results, but at least five insecticide treatments showed promise in limiting virus spread during spring 2008. Increasing concern among growers in this region regarding recent epidemics of CYSDV is leading to more intensive use of insecticides that threatens to erupt into unmanageable resistance. Sustainability of insecticides is an important goal of pest management and more specifically resistance management, especially for some of the most notorious vector species such as *B. tabaci* and *Myzus persicae* that are likely to develop resistance.

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### 1. Introduction

Management of vector-borne plant diseases has long represented an enormous challenge due to the complex dynamics and interactions of host plants, vectors and viruses within a variable environment. These three major components of the vector 'disease triangle' are subject to an indeterminable interplay of physical and biotic factors that influence disease trajectories within crops. Understanding how these factors affect disease incidence and implementation of both proactive and reactive measures to mitigate its occurrence is the goal of applied plant virus epidemiology. However, basic knowledge about epidemics of vectored viruses is often lacking to the extent that the component parts are seldom fully elucidated. For example, identification of host plant reservoirs of a virus outside of the crop is notoriously lacking for many pathosystems (Racchah et al., 1988), thus impeding management solutions that might otherwise be directed at eliminating virus reservoirs. Similarly, uncertainty about the identity of vector species, the efficiency with which various species transmit virus, or the relative numbers of the various vector species moving into and potentially colonizing a crop all reflect a fundamental lack of knowledge about what drives virus epidemics and how they should

be managed. Conceptually, these various aspects of the vector–virus interaction all integrate into the term 'vector-intensity' (Irwin and Ruesink, 1986). This, along with other terminology used to describe interactions among components of the disease triangle, are invaluable for organizing thinking about the problem of vector-borne viruses. However, crop protection practitioners require more than a concept to approach the problem – effective management can benefit most by the fullest elaboration of the parts and interactions of a pathosystem in conjunction with identification of the most effective control measures to reduce virus spread.

In seeking to protect annual crops from the debilitating effects of viruses, growers are confronted with the dilemma that interactions outside of the crop that result in the primary spread of virus into the crop are generally beyond individual control. Although a similar situation faces the grower concerning immigration of a non-vector pest population into an emergent crop, adherence to integrated pest management (IPM) practices enables close monitoring of incipient infestations in the crop and advises treatment action upon reaching a predetermined economic threshold (Stern et al., 1959). So long as effective treatments are available to suppress non-vector pest infestations below economic injury level, lingering concerns about the potentially harmful status of an infestation can be addressed in real time and handled by further treatments if necessary. In other words, what is seen and how timely and effective the reaction generally determines the outcome of non-vector pest population suppression.

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By contrast, much greater effort is required to monitor what often amounts to minimal densities of a transient vector population moving across a field and spreading virus. In most cases, traps for monitoring vector flight are beyond the scope of what most growers are willing to invest to protect their crops, especially in situations where vector-borne diseases occur inconsistently from 1 year to the next. Hence, awareness of vector presence usually does not come until the first plants begin to show disease symptoms. This situation becomes more critical in that during the period of latency when virus replication and systemic spread within a plant is occurring before symptoms appear, sources of inoculum within the crop are likely to have been available as foci for secondary spread. If this is so, then both primary spread and the beginning of secondary spread may have occurred before a grower sees the first symptoms and can react to the incipient epidemic. Even then, however, there are no simple remedies such as a spray application to cure an infection in the same way that an arthropod pest infestation can be cured. At this point the grower may consider sending a roving crew into the field to eliminate diseased plants, but has to be concerned about the ability of the crew to spot a diseased plant and differentiate from other unusual symptoms not related to virus infection, not to mention the high cost of labour relative to a spray application. In short, the visual data required to make educated decisions about treating a vector population, especially one vectoring a non-persistent virus, is often deficient due to an inherent information lag that is absent from an IPM-based system for a non-vector pest. Similarly, options for dealing with infected plants in the field are limited relative to those available for managing a non-vector pest.

Despite the inherent difficulties associated with suppressing vector-borne viruses, the management situation has improved considerably in recent years with the advent of safer and more effective insecticides (Casida and Quistad, 1997). Many newer modes of action have been discovered and developed commercially that support the trend towards greater selectivity in the chemical treatments used in IPM programmes. Although primarily developed for the purpose of controlling various insect pests, including those that vector viruses, a secondary benefit in many cases has been the impact that certain treatments have on inhibiting disease progress. The benefit that results is often more than might be expected simply as an outcome of suppressing vector populations and significantly reducing the number of individuals available for transmission of the virus. Some of these compounds show distinctive antifeedant properties at sublethal doses, while others appear to exert a kind of paralysis to the mouthparts of sucking insects. More precise understanding of the behavioural effects that various compounds have on feeding mechanisms of vectors might enable more expert use of chemical treatments in IPM programmes that especially target disease prevention.

A good example of the dual benefit exhibited by particular insecticides has been seen in IPM programmes developed for the sweet potato whitefly, *Bemisia tabaci* Gennadius. This highly polyphagous insect infests crops worldwide and is known to transmit over 110 viruses (Jones, 2003). In some areas its greatest impact has been during severe outbreaks resulting in direct feeding damage to various crops. One such area is the southwestern USA where intensive year-round agriculture common to the irrigated desert valleys has provided near-optimal conditions for the heat-tolerant *B. tabaci*. Outbreak conditions during the early 1990s severely decreased productivity in the Imperial Valley of California (Gonzalez et al., 1992; Perring et al., 1993) and other growing regions extending into Arizona. More recently, however, viruses previously unknown to this region and transmitted by *B. tabaci* have become established and are affecting the productivity of vegetable and melon crops. These viruses include *Lettuce chlorosis virus* (McLain et al., 1998), a closterovirus of the genus *Crinivirus* that was first detected in the early

1990s in California in conjunction with the displacement of the indigenous *B. tabaci* biotype A by the now predominant *B. tabaci* biotype B (= *B. argentifolii* Bellows & Perring). In cantaloupes (*Cucumis melo* L.), *Cucurbit leaf curl virus* (CuLCV) is a whitefly-transmitted geminivirus first observed in the southwestern USA and northern Mexico between 1998 and 2000 and now found commonly in cucurbits (Brown et al., 2002). The latest discovery in this region is *Cucurbit yellow stunt disorder virus* (CYSDV), another *Crinivirus* that has become prevalent in cantaloupe fields in recent seasons and is causing much concern among growers (Kuo et al., 2007). CYSDV is transmitted semipersistently by *B. tabaci* and is well known from Mediterranean countries, especially in areas such as southern Spain where there is intensive vegetable and melon production and chronic infestations of *B. tabaci*. Prior emphasis in the southwestern USA on controlling *B. tabaci* populations to prevent damage caused by direct feeding has now broadened to include measures that can effectively prevent or significantly delay virus transmission and disease onset.

This paper will focus on newer chemical treatments that are already being used or are in development against *B. tabaci* with respect to their ability to manage infestations as well as control virus disease incidence. Results for other vector species and the impact on associated virus diseases using the same or related chemical treatments will also be reviewed, but with an emphasis on insecticides developed in the past 20 years. The modes of action of these various insecticides will be discussed in relation to how they may disrupt virus transmission and mitigate disease incidence with respect to mode of transmission by insect vectors.

## 2. Materials and methods

### 2.1. Field experiments

Cantaloupes are typically produced in the southwestern USA during spring and autumn growing seasons which correspond to early and peak development of *B. tabaci* populations, respectively. Profound differences in vector pressure between the two seasons occur as continuous immigration of adult *B. tabaci* during autumn into the fields of newly emerged cantaloupe plants often results in hundreds of adults per plant at the single true-leaf stage. In contrast, immigration pressure is much less during spring as *B. tabaci* populations are gradually rebuilding following cool winter months. Consequently, management of *B. tabaci* populations and CYSDV incidence in cantaloupe fields is a much greater challenge in autumn than in spring.

### 2.2. Systemic insecticide trial

Field trials were carried out during both growing seasons at the University of Arizona Yuma Agricultural Center to evaluate the performance of various insecticide treatments against *B. tabaci* infestations and incidence of CYSDV. During the 2007 autumn season, cantaloupe plots were direct-seeded on 16 August with cv 'Gold Express' into two 2.1 m × 15.2 m beds. A 2.1-m buffer between plots provided separation between treatments. The study was designed as a randomized complete block design with four replicates per treatment. A total of four systemic insecticide treatments were applied that included the following: (1) Durivo® at 156 ml ha<sup>-1</sup>, a soluble concentrate containing 0.3 kg a.i. l<sup>-1</sup> in a 2:1 ratio of thiamethoxam + chlorantraniliprole; (2) chlorantraniliprole at 80.3 ml ha<sup>-1</sup>; (3) thiamethoxam at 102 ml ha<sup>-1</sup>; (4) imidacloprid at 215.7 ml ha<sup>-1</sup>; and an untreated check. All treatments were applied to the soil and placed 7.6 cm below the seedline prior to planting at 33.71 ha<sup>-1</sup>. No additional insecticide treatments were applied to the cantaloupe crop.

**Table 1**

Rates of foliar insecticides used on Spring 2008 cantaloupes in Yuma, AZ.

Treatment	Rate (ml ha <sup>-1</sup> )	
	Compound	
	1	2 <sup>a</sup>
Endosulfan + pymetrozine <sup>a</sup>	33	384
NNI-0101	72	–
Cyazapyr 0.83SE	240	–
Chlorantraniliprole	84	–
Dinotefuran + chlorantraniliprole <sup>a</sup>	84	48
Volium Flexi	84	–
Spirotetramat	60	–
Dinotefuran + bifenthrin <sup>a</sup>	48	60
Endosulfan + bifenthrin <sup>a</sup>	384	60
Dimethoate + bifenthrin <sup>a</sup>	288	60
Untreated control	–	–

<sup>a</sup> Mixture of two compounds.

### 2.3. Foliar insecticide trial

Ten foliar spray treatments (Table 1) were tested in spring cantaloupes planted on 14 May 2008 and grown as in the autumn trial. The experimental plots were arranged in a randomized complete block design with four replications per treatment. The foliar spray treatments were applied with a CO<sub>2</sub> backpack sprayer that delivered 235 l ha<sup>-1</sup> at 3.5 kg cm<sup>-2</sup>, using 3-TX18 ConeJet nozzles per bed. Foliar applications were made on 2, 10 and 24 June. The numbers of assessments of adult densities following each of the three applications were 2, 4, and 3, respectively. All spray treatments included the adjuvant DyneAmic at 0.35% (v/v).

### 2.4. Estimation of whitefly densities

Different approaches were used to determine the impact of insecticide treatments on densities of *B. tabaci* in the two trials. The intense immigration pressure in the autumn trial precluded meaningful adult counts. Therefore, whitefly immature densities were estimated by sampling two leaves on the primary vines (terminal and crown leaves) from each of five plants in each plot. Eggs and nymphs were counted on 2 cm<sup>2</sup> leaf disks on each leaf using a dissecting microscope. Immature densities were averaged across leaf positions on each sample date and reported as immature numbers per leaf disk. In the spring trial, populations of whitefly adults were evaluated at various intervals following each application. Adult populations were estimated by taking leaf turn samples from the fifth terminal leaf on the primary melon vine of 10 randomly selected plants per replicate.

### 2.5. Estimation of CYSDV incidence

Incidence of CYSDV was estimated by counting the number of leaves in each 13.7 m plot that expressed both early symptoms of pale leaf discoloration and more severe yellowing of leaf tissue showing obvious interveinal chlorosis (IVC). The number of leaves on mainstem branches expressing symptoms was counted for each plant included in the survey. Multiple leaf samples showing representative symptoms were collected each season and confirmed for the presence of CYSDV in Dr. Judy Brown's Laboratory (University of Arizona, Tucson, AZ, USA) using PCR detection methods.

### 2.6. Greenhouse experiments

The capacity of the neonicotinoid insecticide imidacloprid to prevent transmission of CYSDV was investigated under controlled conditions in the greenhouse. Cantaloupe test plants at the one true-leaf stage were treated with either a high (6 mg a.i. per

container) or low (4 mg a.i. per container) concentration of imidacloprid, or with water as a control 72 h prior to exposure with *B. tabaci* adults. Following treatment and systemic uptake, test plants were randomly allocated to three different test groups to be challenged by viruliferous whiteflies. Experimental whiteflies were aspirated from a colony hosted on cotton plants and confined within a whole-leaf cage enclosing a CYSDV-symptomatic cantaloupe leaf for a 48 h acquisition access period. Cantaloupe test plants were covered within ventilated plastic cups (0.7 l capacity) with corked portals to enable 3, 10, or 30 whiteflies to be introduced to each enclosed plant. Test plants were maintained under grow lights for a 48 h inoculation access period (IAP) and then evaluated to determine the number of surviving whiteflies on the imidacloprid-treated and control plants. After evaluating mortality and removing all surviving whiteflies, test plants were relocated to an insect-free greenhouse, sprayed with bifenthrin insecticide to eliminate residual whiteflies and held for 21 days to allow symptom development. Ten days after the IAP, two 1.5 cm leaf disks were punched from each test plant to evaluate imidacloprid concentration. An Envirologix® (Portland, ME, USA) test kit (EP 006) that employs a competitive ELISA procedure was used to quantify imidacloprid concentrations in leaf samples with a limit of detection of 0.2 ng ml<sup>-1</sup>.

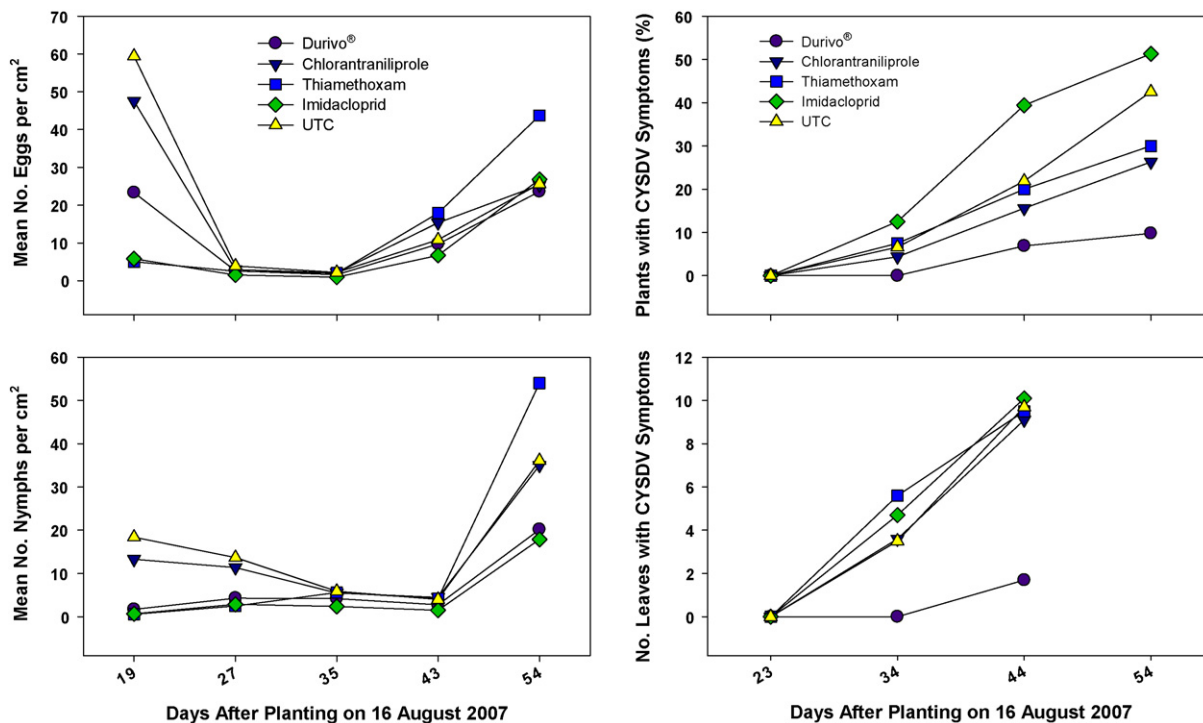
## 3. Results

### 3.1. Autumn 2007 field trial

Heavy immigration pressure into the cantaloupe plots was apparent from the high egg densities recorded on 4 September, especially in the untreated control and chlorantraniliprole plots (Fig. 1). Egg densities on older leaves remained low over the next two sampling dates as adult whiteflies shifted feeding and oviposition to younger leaves at branch terminals. Both egg and nymph densities increased in the late season, perhaps in conjunction with the first in-field generation of *B. tabaci* emerging as adults and establishing a resident population of adults rather than simply an itinerant immigrant population. Although egg and nymphal densities remained suppressed in treated plots through much of September, incidence of CYSDV increased in all treatments, but at variable rates. The imidacloprid treatment was least effective based on the greater incidence of CYSDV than even the untreated control (Fig. 1). The Durivo treatment provided the best protection against CYSDV incidence even though *B. tabaci* egg and nymph densities were similar to other treatments on most of the sampling dates. The reduced incidence of CYSDV in Durivo-treated cantaloupes was also manifested by the fewer number of leaves showing CYSDV symptoms compared to other treatments (Fig. 1).

### 3.2. Spring 2008 field trial

The gradual buildup of whiteflies that occurs in the southwestern US that begins with warmer weather in the spring was evident in the various treatment plots. In early June, less than 10 adult *B. tabaci* per leaf on average occurred in all plots including the untreated control (Fig. 2). By mid-June, the average number per leaf was increasing rapidly in the untreated control plots, whereas most of the treatments showed some level of reduction in adult numbers. Treatment differences really began to show in late June as whitefly infestations continued to increase as evidenced by high numbers in the untreated control plots. The third application on 24 June of certain insecticide treatments helped to maintain moderate numbers of adults despite increasing whitefly pressure. Among the best performers were the two compounds known to physiologically affect feeding by aphids and whiteflies, although the target site for neither compound is unknown. These compounds are the pymetrozine + endosulfan treatment in which pymetrozine is the

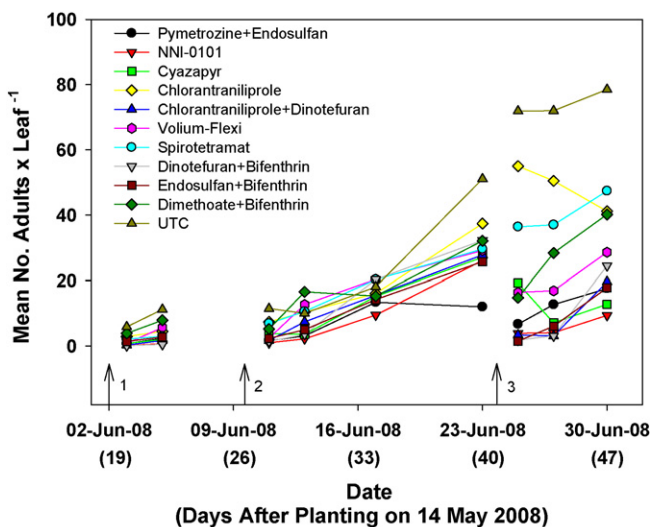


**Fig. 1.** Mean number of *B. tabaci* eggs and nymphs per cm<sup>2</sup> of cantaloupe leaf tissue for each of four soil-applied, systemic insecticides (applied on day of sowing, 16 August 2007) and the untreated control (UTC). The incidence of CYSDV was recorded as the relative proportions of plants and the number of leaves per plant showing CYSDV symptoms.

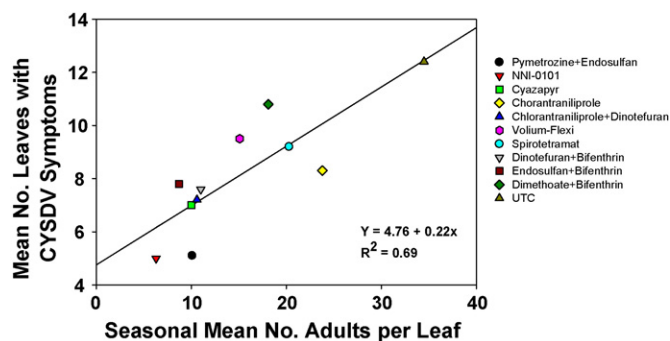
ingredient that affects the feeding mechanism, and the experimental compound pyrifluquinazon (NNI-0101, Nichino) that is still under development. Also performing well were the cyazapir treatment which acts on the ryanodine receptor in muscle and nerve tissue, the mixture of dinotefuran + chlorantraniliprole, and the older compounds represented by the endosulfan + bifenthrin treatment (Fig. 2). Plants receiving these treatments all had fewer leaves showing CYSDV symptoms and comparatively fewer adult whiteflies than the remaining treatments including the untreated control (Fig. 3). A significant regression ( $F_{2,11} = 20.1$ ,  $P = 0.0015$ ) indicated the positive relationship between adult numbers ( $Y$ ) and CYSDV severity ( $x$ ) as described by the equation:  $Y = 4.76 + 0.22x$ ,  $R^2 = 0.69$ .

### 3.3. Greenhouse transmission tests

The amount of CYSDV transmission to potted cantaloupe plants treated with the lower dose of imidacloprid actually exceeded that of the untreated control plants (Fig. 4A). However, the higher dose provided some protection as virus infections were fewer at the 10 and 30 adult infestations, although differences among the three treatments (including untreated control) were non-significant ( $\chi^2 = 7.8$ ,  $df = 4$ ,  $P = 0.099$ ). All plants recorded positive for CYSDV expressed symptoms within 10–14 days following the IAP. At the three-adult density, 4–12 plants became infected in the high dose treatment compared to 3 of 12 in the low dose treatment or the untreated control plants. A possible explanation for this can be seen in the wide variation in imidacloprid concentration among the 12 plants treated with a high dose of imidacloprid and assigned to the three-adult density (Fig. 4B). Although treated with the higher concentration of imidacloprid, 9 of the 12 plants had tissue concentrations of imidacloprid that were equivalent to the plants treated

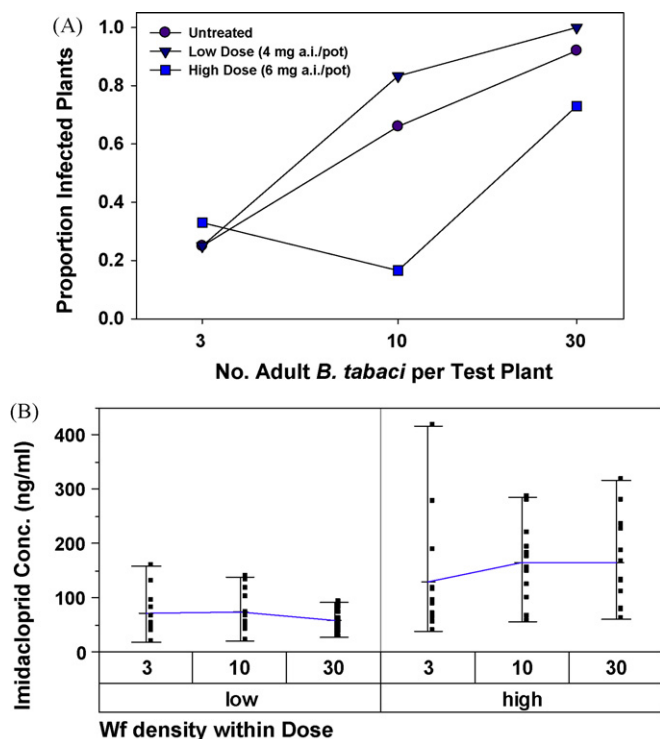


**Fig. 2.** The impact of 10 foliar insecticide treatments on mean number of *B. tabaci* adults per leaf after three applications (see arrows) of each treatment.



**Fig. 3.** Positive relationship between seasonal mean number of adults per leaf and mean number of leaves on the primary branch with CYSDV symptoms; based on differential numbers of adults according to insecticidal treatment.





**Fig. 4.** Transmission of CYSDV by *B. tabaci*: (A) at three densities (3, 10, 30) to untreated control plants and to test plants treated with a high or low rate of imidacloprid or left untreated as control plants, and (B) concentrations of imidacloprid in leaf punches taken from test plants. The range of concentrations for each application rate of imidacloprid and at each whitefly density is delimited by the vertical lines, while the traversing line intersects with the vertical line at the mean concentration for each treatment group.

with the low dose and thus were more vulnerable to feeding and transmission of CYSDV. The mean concentration of imidacloprid in the high rate treatment was  $153 \pm 15 \text{ ng ml}^{-1}$  compared to the mean of  $67 \pm 5.8 \text{ ng ml}^{-1}$  for the low rate treatment ( $N=36$  for each treatment).

## 4. Discussion

### 4.1. *B. tabaci* and CYSDV

The problems that often confront growers in managing insect-vectored virus diseases are well exemplified by the *B. tabaci*/CYSDV pathosystem in cantaloupe-producing areas of Arizona and California. The large numbers of *B. tabaci* observed in spring and especially in the autumn growing seasons increase the probability of virus transmission provided that inoculum sources are numerous and nearby a cantaloupe field. As a highly polyphagous feeder, *B. tabaci* develops on many host crops, ornamentals and weeds that are beyond the direct control of individual growers. The importance of early crop destruction and good sanitation to prevent weeds and other host plants from contributing to the buildup of regional *B. tabaci* populations has been stressed often to growers and pest control practitioners. However, in the absence of area-wide control programmes, prevention of vector immigration and primary spread of virus into a crop, as well as prevention of colonization and secondary spread of virus within that crop, becomes a matter of local control by individual growers without the potential benefit of a coordinated control programme.

The incomplete characterization of the *B. tabaci*/CYSDV pathosystem in the southwestern USA has thus far placed a tremendous reliance on insecticides to combat virus spread. Research is

progressing to identify reservoir hosts for CYSDV so that additional measures might be taken to avoid epidemics. The complete breaks in cucurbit plantings that occur in July between the spring and autumn growing seasons and again in December and January between the autumn and spring seasons make it clear that alternative hosts are involved in the ecology of CYSDV. Cantaloupe fields in this region are always direct-seeded at the time of field planting, so there is no possibility that primary inoculum sources reach the fields as transplants. It is therefore crucial to determine which of potentially many plant species are serving as reservoirs for CYSDV and the nature and level of interaction with *B. tabaci* for each species. Although easily stated, this type of investigative research can easily require years of patient and often frustrating attempts to identify the components and interactions of the pathosystem and to enable cost-effective measures to control disease incidence to be implemented.

Meanwhile, growers face the enormous challenge of preventing the initial establishment and subsequent spread of CYSDV through the use of insecticides and/or barriers such as row covers. As many as 13 generations of *B. tabaci* per year occur in the southwestern USA (Palumbo et al., 2001) with peak populations observed in late summer and early autumn just as newly planted cantaloupes and other vegetable crops are germinating. The intense pressure from adults confronting young plants was evident during the autumn 2007 season when a mean of 60 *B. tabaci* eggs per  $\text{cm}^2$  of cantaloupe leaves occurred in the untreated control plots on 4 September only 19 days after sowing. Although the neonicotinoid treatments imidacloprid and thiamethoxam prevented such high densities of eggs and nymphs so early in the season, they were relatively ineffective at suppressing incidence of CYSDV relative to the untreated control and the other two insecticide treatments. The incomplete suppression of CYSDV by the two neonicotinoids could have multiple explanations, but a strong possibility may be that resistance to these compounds (Prabhaker et al., 2005) has built-up after more than a decade of intensive use in the Yuma region. Resistance to neonicotinoids might enable longer acquisition and inoculation access feedings and promote higher rates of transmission compared to a susceptible vector population. In contrast to the poor performance of thiamethoxam alone, the combination of thiamethoxam with chlorantraniliprole in the commercial formulation known as Durivo provided relatively good control of CYSDV in terms of the proportion of plants as well as the number of leaves on each infected plant showing CYSDV symptoms. Many growers of autumn season cantaloupes have become resigned to merely delaying the onset of CYSDV infection rather than total prevention. The intensity and constancy of *B. tabaci* pressure during autumn is simply too great to achieve this. Cantaloupe fruit quality has declined in recent years due to the detrimental effect of CYSDV on accumulation of sugars (Celix et al., 1996; Sinclair and Crosby, 2002). Vines that remain free of CYSDV for a longer time period have a better chance of producing marketable fruit of good quality compared to an early infected plant.

The concern about CYSDV epidemics is not as great in spring season cantaloupes due to the much lower vector-intensity relative to autumn. Nevertheless, *B. tabaci* is especially attracted to cantaloupe fields and populations build up rapidly if preventive action is not taken. Foliar insecticide treatments can be applied at any time during the season in response to a *B. tabaci* infestation, whereas the soil-applied treatments are most effective early in the crop phenology when applied at the time of planting. At least five different insecticide treatments were effective in reducing the number of adults per leaf and delaying infection by CYSDV. The positive relationship between the mean number of adults per leaf and mean number of leaves per plant expressing CYSDV symptoms was revealed because of the differential efficacies of the 10 foliar insecticides against *B. tabaci* adults. However, it is obvious from

the high Y-intercept value of 4.76 in the regression equation that spot counts of *B. tabaci* adults only provide an index of comparison among the various treatments, but do not tell the full story of exposure that occurs from day to day. Otherwise, the regression line would be expected to pass through the origin without the additional occurrence of almost five leaves per plant with CYSDV symptoms. These additional leaves represented in the Y-intercept value probably result from the cumulative exposure and feeding by *B. tabaci* adults that are not recorded when only periodic counts are made.

Evaluation of insecticides in the field against whitefly infestation and virus transmission provides a real test of efficacy, but only within the context of the conditions during a particular test. For example, comparison of one treatment for control of CYSDV during the spring season with another tested during the autumn is unrealistic because of the big difference between seasons in vector-intensity. Consequently, testing of multiple products or different rates of a single product under standardized conditions in the greenhouse provides a means of comparing efficacies across multiple tests. In our study, the higher rate of imidacloprid provided modest protection against CYSDV compared to the lower rate or the untreated control plants. However, there were considerable differences in systemic uptake of imidacloprid between test plants in the respective treatment groups despite adopting a standard treatment protocol. Variation in root growth and density in the soil within each test pot was likely to be the reason for differential uptake. The mean concentrations of imidacloprid attained in both the high and low rate test groups were somewhat less than concentrations that have been measured in field-grown and treated plants. Imidacloprid concentrations in field-grown cantaloupe leaves of 2–4-week-old plants often range from 200 to 450 ng ml<sup>-1</sup> (S. Castle and J. Palumbo, unpublished), so a higher dose of imidacloprid will be included in future greenhouse tests to better duplicate concentrations observed in the field.

#### 4.2. Insecticidal control of plant viruses

Given the paucity of viable management tactics to prevent vector-borne disease, growers often seek to protect their crops by targeting the vector component with insecticides. Perring et al. (1999) explained that growers in California and the US understand that vector-borne crop diseases are frequently caused by viruses and not the insects that vector them. They also described various reasons why growers often rely upon insecticides to prevent infections. To what degree growers understand differences in transmission mode, primary vs. secondary spread, and the mode of action of insecticides used to prevent the spread of viruses in their crops is uncertain, but all are highly relevant to the overall success of the prevention strategies adopted.

As generally with insecticides used to control pest populations, greater knowledge of the modes of action and activity profiles of candidate insecticides will improve opportunities for controlling vector populations and mitigating transmission of virus from one plant to another. The list of options for selecting a particular insecticide treatment has expanded considerably in recent years as newer and more selective modes of action have been developed and commercialized. Many of these compounds may affect disease incidence only through toxic activity against the vector population and not necessarily through antifeedant properties or other mechanisms that affect behaviour and disrupt transmission. For *B. tabaci*, there are now numerous compounds representing some 10 different modes of action that are effective in controlling infestations (<http://www.irac-online.org/documents/moa.whiteflyposter.pdf>). These compounds helped to reduce outbreak conditions that existed in the southwestern USA during the 1990s (Palumbo et al., 2001).

Among the most significant developments in pesticide chemistry of the last 20 years has been the discovery and expansion of neonicotinoid insecticides. The target site within the insect for compounds of this class is the nicotinic acetylcholine receptor (nAChR). Imidacloprid was the first such compound to be commercialized (Leicht, 1993) and is the one for which the most abundant information has been generated regarding impact on virus transmission (Table 2). In addition to being very effective in suppressing populations of many hemipteran vector species, imidacloprid is also well known for possessing distinctive antifeedant properties against the aphid vector *Myzus persicae* and *B. tabaci* (Nauen, 1995; Nauen et al., 1998a,b). At least one other related compound, thiamethoxam, is also effective in reducing certain viral diseases (Table 2), but so far no evidence has been produced that antifeedant or disruptive mechanisms other than toxicity to the insect are involved (Mowry, 2005). Systemicity is another property of imidacloprid and most other neonicotinoids that allows considerable flexibility in how the treatment is applied to the crop. A seed-coating of imidacloprid or application to the furrow at planting enables protection of seedlings as they emerge from the soil. Imidacloprid can also be applied through drip irrigation systems and delivered direct to the root zones of plants for efficient uptake. When applied as a systemic treatment the chemical has long residual activity that helps protect against sustained insect pressure (Palumbo et al., 2001). Additional formulations of imidacloprid and other neonicotinoids make possible foliar treatments that can be applied in response to infestations as they reach economic thresholds.

Pymetrozine is another newer insecticide that has antifeedant properties, but the mode of action is unclear (Table 2). This insecticide is particularly effective against certain aphid species by arresting feeding behaviour to such a degree that affected aphids eventually starve to death (Harrewijn and Kayser, 1997). Pymetrozine has also been shown to be effective at preventing virus transmission by *B. tabaci* (Polston and Sherwood, 2003). The experimental compound pyrifluquinazon (NNI-0101) tested against *B. tabaci* field transmission of CYSDV in the current study has also demonstrated strong antifeedant properties and continues to be examined for its potential to disrupt virus transmission.

There are many examples of older insecticides that are effective in reducing viral disease incidence (see reference in Perring et al., 1999). Many of these compounds, including the pyrethroid bifenthrin and the cyclodiene endosulfan used in our study against *B. tabaci*, continue to prove valuable in helping growers to curb losses due to vector-borne viral diseases. In situations such as the *B. tabaci* infestations in the southwestern USA, insecticide mixtures may prove necessary by combining compounds with antifeedant or repellent properties with toxic compounds having lethal activity to help suppress populations. In other areas where vector-intensity is not as high, more finessed approaches that take advantage of the unique properties of certain insecticides such as the disruption of aphid feeding by pymetrozine (Harrewijn and Kayser, 1997) may prove adequate for minimizing viral disease incidence in crops.

#### 4.3. Dangers of resistance

Realistic concern about epidemics of vector-borne viruses in crops has raised the stakes in terms of the consequences of intensified insecticide use. The inability to accurately forecast virus incidence in most pathosystems leaves growers little recourse other than to protect their crops aggressively with frequent use of insecticides against vectors that are often transient and difficult to monitor. In areas such as southern Spain where growers have long fought against *B. tabaci*-vectored viruses, high levels of resistance to imidacloprid and other insecticides have been observed in *B. tabaci* populations (Nauen et al., 2002; Nauen and Denholm, 2005). In developing countries of Latin America, Africa,

**Table 2**

Examples of three insecticide compounds that have proven effective against various vectors and the virus diseases they spread.

Vector		Virus		References	Evidence
Taxonomic group	Species	Species	Transmission mode		
(A) Compound: imidacloprid; chemical subgroup: neonicotinoid; mode of action: nicotinic acetylcholine receptor agonist/antagonist					
Aphids, Hemiptera, Aphididae	<i>Myzus persicae</i>	<i>Beet mild yellowing virus</i> (BMVY)	Persistent	Qi et al. (2004) Boiteau and Singh (1999), Mowry (2005), Mowry and Ophus (2002) Dewar et al. (1992) Gray et al. (1996), McKirdy and Jones (1996, 1997), Gourmet et al. (1996), Wangai et al. (2000)	Field & Lab
		<i>Potato leafroll virus</i> (PLRV)	Persistent		
		<i>Beet yellows virus</i> (BYV)	Semipersistent		
Thrips, Thysanoptera, Thripidae	<i>Rhopalosiphum maidis</i>	<i>Barley yellow dwarf virus</i> (BYDV)	Persistent	Riley and Pappu (2004), McPherson et al. (2002, 2003), Coutts and Jones (2005)	Field
	<i>R. padi</i>				
	<i>Frankliniella occidentalis</i>				
	<i>F. fusca</i>	<i>Tomato spotted wilt virus</i> (TSWV)	Persistent propagative		
	<i>F. bispinosa</i>				
Whitefly, Hemiptera, Aleyrodidae	<i>F. schultzei</i>			Sreekanth et al. (2003, 2004) Ahmed et al. (2001) March et al. (2002)	Field Field Field & Lab
	<i>Thrips tabaci</i>				
	<i>T. palmi</i>	<i>Peanut bud necrosis virus</i> (PBNV)	Persistent		
Planthopper, Hemiptera, Delphacidae	<i>Bemisia tabaci</i>	<i>Tomato yellow leaf-curl virus</i> (TYLCV)	Persistent	Wang et al. (1999)	Field & Lab
	<i>Delphacodes kuscheli</i>	<i>Mal de Rio Cuarto virus</i> (MRCV)	Persistent propagative		
Leafhopper, Hemiptera, Cicadellidae	<i>Circulifer tenellus</i>	<i>Beet curly top virus</i> (BCTV)	Persistent		
(B) Compound: thiamethoxam; chemical subgroup: neonicotinoid; mode of action: nicotinic acetylcholine receptor agonist/antagonist					
Aphid	<i>M. persicae</i>	PLRV	Persistent	Mowry (2005)	Lab
Thrips	<i>F. occidentalis</i>			McPherson et al. (2002, 2003), Coutts and Jones (2005)	Field
	<i>F. fusca</i>				
	<i>F. bispinosa</i>	TSWV	Persistent propagative		
	<i>F. schultzei</i>				
Whitefly	<i>T. tabaci</i>			Mason et al. (2000)	Lab
	<i>B. tabaci</i>	TYLCV	Persistent		
(C) Compound: pymetrozine; chemical subgroup or active ingredient: pymetrozine; mode of action: compounds of unknown action or nonspecific modes of action (selective feeding blockers)					
Aphid	<i>M. persicae</i>	PLRV	Persistent	Mowry (2005)	Lab
Whitefly	<i>B. tabaci</i>	TYLCV	Persistent	Mason et al. (2000)	Lab

India and southeast Asia, *B. tabaci* is an efficient vector of plant viruses affecting important food crops (Morales, 2005), particularly common bean (*Phaseolus vulgaris* L.) (Morales and Anderson, 2001), cassava (*Manihot esculenta* Crantz) (Fargette et al., 1990; Gibson et al., 1996), tomato (*Lycopersicon esculentum* Mill.) (Polston and Anderson, 1997) and peppers (*Capsicum* spp. L.) (Morales and Anderson, 2001). Excessive applications of insecticides made in these regions to combat infestations of whiteflies and to prevent the endemic virus diseases often lead to resistance and a reduced arsenal of effective insecticides (Morales, 2005). In Colombia and Ecuador, over-reliance on insecticides for whitefly control has been so widespread that 30% of 325 farmers interviewed reported making more than 10 applications per cropping season (Cardona et al., 2005). Similarly, resurgence of *B. tabaci* in cotton and vegetables over large areas of India during the 1980s was attributed to the “indiscriminate use of insecticides” (Sundaramurthy, 1992).

The reaction to the onset of *B. tabaci*-transmitted viruses in California and Arizona has been an intensification of chemical management to avoid the quality-diminishing effects of CYSDV. In many situations, growers are applying neonicotinoid (as well as endosulfan and bifenthrin) sprays to supplement previous soil-applied neonicotinoid treatments. The reduced cost of generic imidacloprid products new to the market in recent years is contributing to increased demand for this valuable insecticide by vegetable and cantaloupe farmers. Due to the appearance of CYSDV, *B. tabaci* populations in the southwestern USA are probably now at the highest risk ever experienced for resistance to neonicotinoids and pyrethroids. One of the potential consequences of intensified insecticide use in vegetable and cantaloupe crops against *B. tabaci* could be a recurrence of the resistance episodes observed in cotton production systems on several continents during the latter part of the 20th century (Dittrich et al., 1985; Prabhaker et al., 1985; Sundaramurthy, 1992; Dennehy and Williams, 1997).

Repeated use of insecticides in response to the constant threat of vector-borne viruses presents a serious difficulty in that selection pressure on the population is raised and resistance risk increased. Insecticide resistance can be a problem in all insect groups that serve as vectors of viral diseases and has appeared in many major arthropod vectors of both medical and agricultural importance. The list of insecticide-resistant vector species includes insects of medical importance such as mosquitoes, body lice, bedbugs, triatomids, fleas and ticks (Anonymous, 1992) and other insects of agricultural importance, such as aphids, whiteflies, and leafhoppers. Of these, various vectors have developed resistance to all classes of insecticides. However, available information on resistance and management of certain vectors of virus diseases shows that the full impact of resistance on control efforts or disease is unknown.

#### 4.4. Resistance and disease control

How significant is the impact of insecticide resistance in vectors on disease control? Generally if the level of resistance is high, vector control is compromised and disease transmission may increase. However, the introduction of new pesticide with different modes of action often mitigates resistance levels and enables better control of vector populations to help prevent disease transmission. The problem with this strategy is that the supply of new modes of action is limited by the number of target sites within the pest organism itself, i.e. there are only so many ways to kill an organism with poison. Resistance management is therefore paramount to the conservation of active ingredients that provide the killing power in pesticides. The erosion of pesticide arsenals due to resistance is a troubling challenge to modern, intensive agriculture that depends heavily on synthetic pesticides to suppress pest populations. Because some of the most notorious vectors of plant viruses, including *M. persicae* and *B. tabaci*, are also among the most com-

mon insecticide-resistant recidivists (Nauen and Denholm, 2005), the stakes for avoiding resistance and maintaining control of vector populations are perhaps even greater than for non-vector insects and must therefore be met with effective pest and resistance management programmes.

Insecticide resistance, whether physiological, biochemical or behavioural, makes vector control problematic. Although mechanisms by which insecticides become resistant are similar across all vector species, each resistance problem can be potentially unique, involving specific changes in target sites or in the structure of particular detoxification enzymes. Regardless of the type of resistance mechanism, the main defence against resistance is to minimize the use of any single mode of action coupled with a close surveillance of the susceptibility of vector populations. Regular monitoring of resistance-prone vector species for susceptibility to commonly used insecticides provides valuable information on resistance severity and for making critical decisions on appropriate insecticides to use to avoid further resistance development.

Regardless of resistance complications, chemical control measures will continue to be relied upon by growers seeking to protect their crops from vector-borne viruses. Previously, the inventory of insecticides available for vector control was reduced by the occurrence of resistance and by removal of those compounds from the market. More recently, however, especially in the past decade, newer insecticides with unique modes of action have become available to control vectors. The capacity to diversify insecticide treatments against *B. tabaci* and other vector species has increased dramatically over the past 10–15 years. At the time of the early 1990s outbreaks of the B-biotype in the USA, treatment options were limited to a selection of conventional materials representing organophosphate, carbamate, pyrethroid and organochlorine insecticide groups. Only three modes of action (MoA) were represented among these four groups as OPs and carbamates both target acetylcholinesterase. This situation has changed dramatically as the number of MoAs available for *B. tabaci* control has increased to 10 according to the most recent update by the Insecticide Resistance Action Committee (<http://www.irac-online.org>). The increasing number of highly effective choices available for pest managers makes possible continuing improvement in management of whiteflies even in the most outbreak-prone locations. Knowledgeable deployment of newer and more selective insecticides will be fundamental to the successful management of insect vectors and the viral diseases they spread.

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